

# EFFECTS OF BLOOD FLOW ON SKIN HEATING INDUCED BY MILLIMETER WAVE IRRADIATION IN HUMANS

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## INTRODUCTION

**Abstract**—We have previously reported species differences in the rate of skin heating in response to millimeter wavelength microwave exposure. We hypothesized that these differences were predominantly a function of species differences in the ability to increase skin blood flow during local heating. Mathematical modeling also suggested that, in humans, the rate of skin heating during prolonged millimeter wavelength exposure would be dependent on skin blood flow. In order to empirically test this hypothesis, we determined the role of baseline skin blood flow on the rate of cutaneous heating induced by 94-GHz microwave energy in humans (3 female, 3 male) using infrared thermography and laser Doppler imaging to measure skin temperature and relative skin blood flow, respectively. Millimeter wavelength exposure intensities used were high power (HP),  $1 \text{ W} \cdot \text{cm}^{-2}$  for 4 s and low power,  $175 \text{ mW} \cdot \text{cm}^{-2}$  for 180 s. Skin blood flow was (a) normal, (b) eliminated using a blood pressure cuff to occlude forearm blood flow, or (c) elevated by heating the skin prior to irradiation. Results showed that for the HP exposures, these manipulations did not influence the rate of skin heating. For the low power exposures, occlusion of baseline skin blood flow had a small impact on the subsequent rate of heating. In contrast, a two-fold elevation in baseline skin blood flow had a profound impact on the subsequent rate of heating, resulting in a substantially lower rate of heating. Occlusion of an elevated skin blood flow reversed this lower rate of heating. The results of these studies demonstrate that relatively small changes in skin blood flow may produce substantial alterations in the rate of skin heating during prolonged 94-GHz exposure.

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**Key words:** electromagnetic fields; radiation, nonionizing; radiofrequency; blood

THE MILLIMETER wave (MMW) frequency range is a subset of the radiofrequency portion of the electromagnetic spectrum, comprising the range of 30–300 GHz. Recently, hardware systems capable of generating radiofrequency radiation (RFR) of MMW length have been developed and are increasingly being used for a number of applications, such as satellite communications, military radar, non-lethal weapons, high speed data communications, automotive anti-collision devices, weapons detection, and medical and dental devices (Moffa et al. 1996; Gilbert et al. 1997; Pakhomov et al. 1998; Carl et al. 2000; Nikawa et al. 2000; Ryan et al. 2000). The increasing use of RF signals in the MMW band in both defense and civilian applications necessitates a better understanding of the bioeffects of electromagnetic energy within this frequency range.

The tissue penetration depth of RF radiation at 94 GHz is approximately 0.4 mm, so the primary bioeffect of such exposure is skin heating (Blick et al. 1997; Ryan et al. 2000). Thus, the use of the specific absorption rate (SAR) to establish safe exposure levels is not appropriate in this frequency band (IEEE 1999). We have recently shown that the rate of skin surface temperature increase in humans in response to brief, high power (HP) MMW exposures can be mathematically modeled using a one-dimensional thermal conduction model (Walters et al. 2000; Nelson et al. 2003). Furthermore, this model is valid across a number of animal species (Nelson et al. 2003). Conduction alone, however, does not adequately explain the dynamics of skin heating during longer, lower-powered (LP) MMW exposures. Although the incorporation of physiologically relevant, steady-state, skin blood flow (SkBF) values into the model explained a portion of the curve, it appeared that SkBF was not at a steady-state, and that alterations in SkBF induced by these longer MMW exposures would significantly alter the subsequent rate of skin heating (Nelson et al. 2003).

In order to further explore this issue, we performed a series of experiments designed to examine the role of

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SkBF in altering the skin heating response to MMW in the human forearm. Forearm SkBF was either increased by local heating or decreased by occlusion of flow to the forearm prior to or during LP MMW exposure, thereby allowing a determination of the influence of SkBF on the heating rate of the skin induced by MMW exposure. Similarly, SkBF was manipulated during (4 s) HP MMW exposures to empirically confirm our previous hypothesis that such exposures may be accounted for by a one-dimensional thermal conduction model.

## MATERIALS AND METHODS

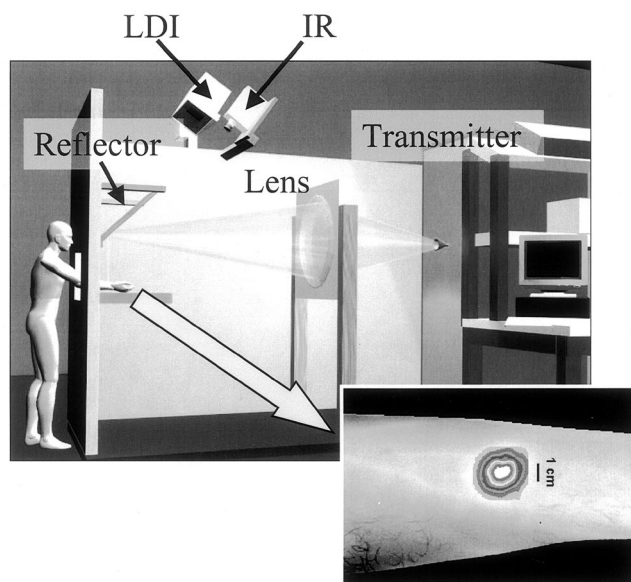
The subjects were three female and three male Caucasian volunteers (military, Department of Defense civilians, and contractors) involved in research on biological effects of RFR. They ranged in age from 25 to 42 y ( $35.8 \pm 2.9$ ; mean  $\pm$  SEM). All subjects were unpaid volunteers who read and signed an informed consent document prior to participation. The use of human subjects in this research was in accordance with a protocol approved by the Institutional Review Board on human use (Brooks Air Force Base, Air Force Research Laboratory Advisory Committee on Human Experimentation) and by the Office of the Surgeon General of the Air Force.

### MMW exposure system

As described previously (Nelson et al. 2003), a 50-W transmitter (Applied Electromagnetics, Inc., Marietta, GA) with a Varian VKB2462L2 gridded extended oscillator emitted microwaves at a frequency of 94 GHz. A Gaussian dielectric lens (Shelton 1991) was used to augment the maximal available power density, by focusing the output of the transmitter into a beam with a diameter of 3.3 cm. The input side of the lens had a focal length of 90 cm, so the lens was positioned 90 cm from the conical output antenna of the transmitter (Fig. 1). Varying the duty factor of a 1,000 pulse/second rectangular wave train that gated the output of the transmitter produced steps in power density below the maximum. A computer program calculated pulse durations necessary to produce the required duty factors (50–90%). A programmable oscillator generated pulse trains with the required duty factors and durations.

### Temperature measurement

Skin surface temperature ( $T_{sk}$ ) was measured continuously before, during and after exposure using infrared (IR) thermography. IR thermography was performed using a Radiance 1 Infrared Camera System (Amber Engineering, Inc., Goleta, CA) equipped with a 50-mm lens. The camera contains a focal plane array composed of  $256 \times 256$  indium antimonide sensors. With the



**Fig. 1.** Schematic of the experimental exposure system. A transmitter emitted 94-GHz microwaves that were then focused by passage through a Gaussian lens. An aluminum block was used to reflect the focused beam onto the subject's forearm. An infrared (IR) camera and laser doppler imaging (LDI) device were positioned to measure  $T_{sk}$  and relative SkBF, respectively. The inset shows infrared thermography of a typical LP exposure on the forearm.

region of interest located 1.0 m from the camera (Fig. 1), each sensor measured the temperature of a square patch of skin  $500 \mu\text{m} \times 500 \mu\text{m}$ . Images were sampled at a rate of either 20 or 0.2 frames per second for HP and LP MMW exposures, respectively. Multi-point calibration with a black-body calibration source (M340, Mikron Infrared, Inc., Oakland, NJ) provided a measured accuracy of  $\pm 0.1^\circ\text{C}$  over the temperature range examined. Automated image analysis was performed off-line using a LabView (National Instruments, Austin, TX) based program. The program determined the mean, minimal, and maximal value of a  $100 \text{ mm}^2$  region of interest.

### Laser Doppler imaging

Relative changes in SkBF were determined using Laser Doppler Imaging (LDI); LDI measures frequency shifts of back scatter photons, which are indirectly related to SkBF (Mack 1998). The LDI device (Moor Instruments, Axminster, UK) was located 1 m from the target area (Fig. 1). Relative SkBF was determined for a region of interest corresponding to that used for IR thermography. For HP, measurements were made just prior to exposure and immediately following exposure. For LP, measurements were made just prior to exposure, at 30-s intervals during exposure and at 30 s following exposure. Scanning speed rate was 15 s per scan of the

1.33 cm<sup>2</sup> region of interest, yielding a scan speed rate of 100 millisecond per pixel and 1.13 pixel per mm<sup>2</sup> resolution. The values reported herein correspond to the mean laser Doppler flux recording for the region of interest. LDI is semi-quantitative and changes in SkBF are therefore reported relative to each subject's baseline value for the normothermic, non-occluded condition.

### Experimental procedures

Room temperature and relative humidity were maintained between 22–24 °C, and 30–50%, respectively. All experiments were performed between 0900 and 1400 h.

The ventral side of the forearm was shaved prior to experimentation. The subject stood outside the exposure chamber and extended his/her arm into the chamber, with the forearm resting on a shelf equipped with a Styrofoam jig designed to minimize movement during exposure (Fig. 1). Each subject completed each of the experimental protocols delineated below over a 2-d period. Each trial requiring preheating was always done after the no-preheating experiment. Exposure parameters (e.g., power density, duration) were randomized within the preheating or no-preheating conditions.

**LP exposures.** The first experimental series was designed to determine how alterations in baseline  $T_{sk}$  and SkBF altered the response to subsequent LP exposure. The ventral side of the forearm was irradiated at a power density of 175 mW cm<sup>-2</sup> for 2 min under conditions of (1) normal baseline  $T_{sk}$  and SkBF (i.e., control conditions); (2) normal baseline  $T_{sk}$  and occluded SkBF; (3) elevated baseline  $T_{sk}$  and elevated SkBF; and (4) elevated  $T_{sk}$  and occluded SkBF. Baseline forearm  $T_{sk}$  and SkBF was elevated by local heating using a commercially available electric heating pad wrapped snugly around the forearm. Heating was applied for 60 min before LP exposure to ensure that a plateau in SkBF was attained. The effectiveness of this treatment in producing an increase and plateau in SkBF was subsequently confirmed in a subset of the subjects using a FLOLAB laser Doppler flowmetry system (Moor Instruments Ltd., Devon, UK) to directly determine SkBF during the heating treatment. Immediately upon removal from the heating pad, the forearm was positioned in the Styrofoam support and the experimental procedure was begun. In experiments with occluded SkBF, a blood pressure cuff was placed on the upper arm and was inflated to 40 mmHg above the individual's systolic blood pressure. Occlusion was performed continuously for 1 min before beginning LP exposure, during the 2-min exposure, and for 1 min following cessation of LP exposure.

A subsequent experiment was designed to determine whether occlusion of SkBF during an extended LP

exposure would alter the rate of skin heating. LP exposure was performed for 3 min to allow  $T_{sk}$  to reach a plateau (as observed in the first experimental series). While continuing the LP exposure, the blood pressure cuff was then inflated and maintained at 40 mmHg above systolic pressure for a 2-min period. At the end of this period, the cuff was deflated and LP exposure continued for a subsequent 3-min period; total continuous LP exposure was therefore 8 min.

**HP exposures.** This experimental series sought to determine whether alterations in baseline  $T_{sk}$  and SkBF altered the skin heating response to short-duration HP exposure (1 W cm<sup>-2</sup> for 4 s). As described above, HP exposure was performed under conditions of (1) normal baseline  $T_{sk}$  and SkBF (i.e., control conditions); (2) normal baseline  $T_{sk}$  and occluded SkBF; (3) elevated baseline  $T_{sk}$  and elevated SkBF; and (4) elevated  $T_{sk}$  and occluded SkBF.

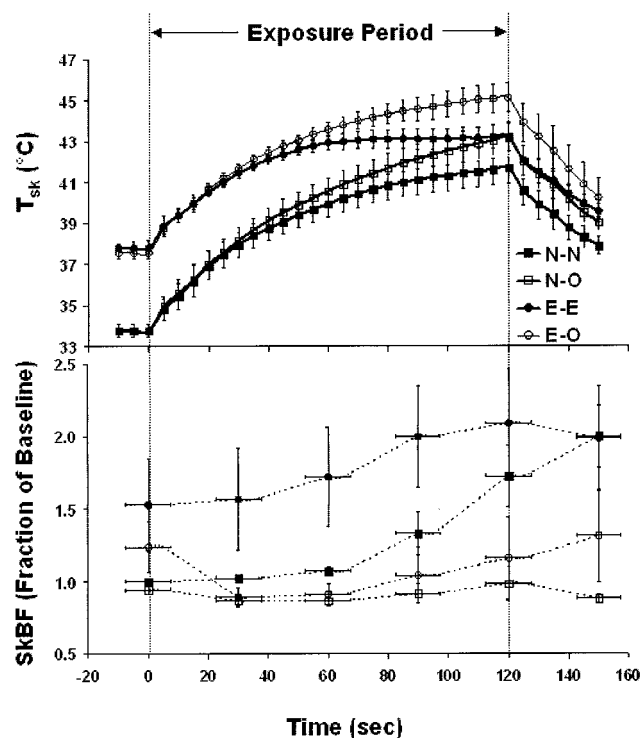
## RESULTS

### LP exposures

Under normal initial conditions in subjects in which SkBF was not occluded, the heating curve produced by LP demonstrates at least 2 phases: an initial rapid phase and a slower secondary phase (Fig. 2). When a heating pad was used to elevate the initial  $T_{sk}$  and SkBF, a third plateau phase was also observed. Examination of the time course of relative increases in SkBF induced by LP exposure reveals that the slower secondary and plateau phases in  $T_{sk}$  temporally correspond with the point at which SkBF increases, as both occur after approximately 60 s of exposure. When blood to the forearm was occluded and SkBF was not allowed to rise in response to LP exposure, a plateau phase was not seen and  $T_{sk}$  continued to increase throughout the exposure period. Preheating of the forearm also decreased the magnitude of change in  $T_{sk}$  during LP exposure under either the non-occluded or occluded SkBF condition.

Based on these observations, a subsequent experiment was undertaken in which SkBF was occluded after  $T_{sk}$  had reached its plateau phase during a 180-s LP exposure. Although this experiment was conducted under initial conditions of normothermia and normal SkBF, a plateau phase was seen in the  $T_{sk}$  response, which again temporally corresponded with the increase in SkBF (Fig. 3); because LP exposure in this experiment was 180 s rather than 120 s as in the first experiment (Fig. 2), this suggests that a plateau phase would have been seen under these same baseline conditions in the first experiment if the duration of LP exposure had been longer. In this experiment, LP exposure increased SkBF approximately





**Fig. 2.** Skin temperature ( $T_{sk}$ ) and skin blood flow (SkBF) responses to LP exposure ( $175 \text{ mW cm}^{-2}$ ) under conditions of (1) normal baseline  $T_{sk}$  and SkBF (i.e., control conditions; N-N); (2) normal baseline  $T_{sk}$  and occluded SkBF (N-O); (3) elevated baseline  $T_{sk}$  and elevated SkBF (E-E); and (4) elevated  $T_{sk}$  and occluded SkBF (E-O). SkBF was normalized to baseline values for the normothermic, non-occluded condition. X-error bars indicate the duration of time required for acquiring a complete image; Y-error bars indicate standard error of the mean.

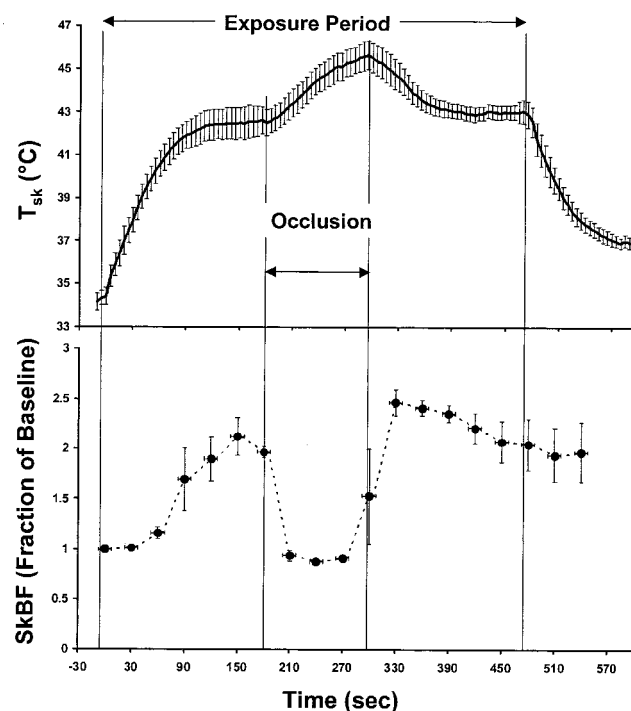
two-fold (Fig. 3). Immediately upon cuff inflation and a return of SkBF to pre-LP exposure values,  $T_{sk}$  increased in a linear fashion; upon cuff deflation and an increase in SkBF,  $T_{sk}$  returned to the plateau level. Taken together, these data indicate that the increase in SkBF induced by local heating during LP exposure decreases the rate of skin heating, resulting in attainment of a steady-state condition in which the energy inflow due to LP exposure is matched by a convective outflow due to a corresponding increase in SkBF.

### HP exposures

Exposure of the forearm to  $1 \text{ W cm}^{-2}$  for 4 s resulted in profound increases in  $T_{sk}$  without altering SkBF (Fig. 4). The rate of  $T_{sk}$  increase was not dependent on either the initial  $T_{sk}$  or the SkBF level, as the responses were similar following preheating and/or occlusion.

## DISCUSSION

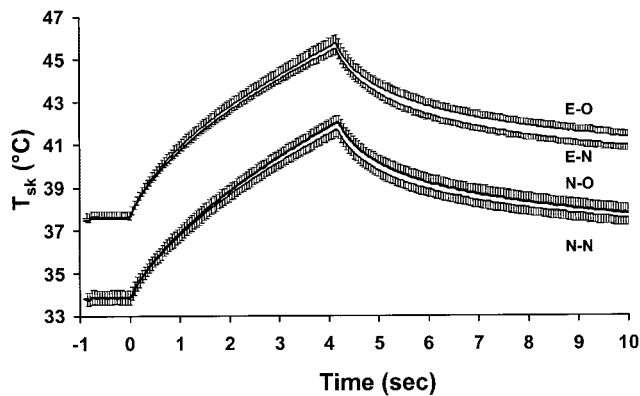
The primary purpose of this investigation was to determine whether manipulations of SkBF could affect



**Fig. 3.** Skin temperature ( $T_{sk}$ ) and skin blood flow (SkBF) during LP exposure. LP exposure ( $175 \text{ mW cm}^{-2}$ ) was initiated under baseline conditions, i.e., ambient temperature =  $22\text{--}24^\circ\text{C}$ . Following 3 min of exposure, a blood pressure cuff was inflated to occlude SkBF. After 120 s of occlusion, the cuff was deflated and SkBF returned. X-error bars indicate range of time at which measurements were made; Y-error bars indicate standard error of the mean.

the rate of skin surface temperature increase during brief HP (4 s) or prolonged LP (2–3 min) MMW exposure. This investigation demonstrates a relationship between surface temperature and SkBF for LP exposure, as the rate of  $T_{sk}$  increase was altered by manipulation of pre-exposure SkBF or by occlusion of SkBF during exposure. Alterations in baseline SkBF did not affect the rate of  $T_{sk}$  increase during HP exposure, confirming our previous hypothesis that the rate of skin surface temperature increase during brief HP exposures could be explained by a one-dimensional thermal conduction model (Nelson et al. 2003).

In a previous study (Nelson et al. 2003), we demonstrated that the increase in  $T_{sk}$  induced by LP exposure differed among species. In the rat and monkey, for example, such exposure produced a biphasic heating curve, in which an initial rapid phase of heating was followed by a slower secondary phase; there was no indication that a plateau was reached using these exposure parameters ( $175 \text{ mW cm}^{-2}$  for 180 s). In the human, however, these two initial phases are followed by a plateau phase, indicating attainment of a steady-state



**Fig. 4.** Skin temperature ( $T_{sk}$ ) and skin blood flow (SkBF) during HP exposure ( $1 \text{ W cm}^{-2}$ ) under conditions of (1) normal baseline  $T_{sk}$  and SkBF (i.e., control conditions; N-N); (2) normal baseline  $T_{sk}$  and occluded SkBF (N-O); (3) elevated baseline  $T_{sk}$  and elevated SkBF (E-E); and (4) elevated  $T_{sk}$  and occluded SkBF (E-O). SkBF was normalized to baseline values for the normothermic, non-occluded condition. Y-error bars indicate standard error of the mean.

condition in which the heat is efficiently removed (this study; Nelson et al. 2003). This observation suggested that, during these longer exposures, SkBF increases due to a direct local heating effect on the cutaneous vasculature (Johnson and Proppe 1996), resulting in an efficient removal of heat by the increased SkBF. Presumably, the noted inter-species variability in the temperature profile induced by MMW heating (Nelson et al. 2003) is therefore due to a greater ability of the human cutaneous vasculature to vasodilate in response to local heating, as humans are the only species studied to date that possess an active vasodilator system and an efficient sweating mechanism over the majority of their surface area (Johnson and Proppe 1996). Furthermore, mathematical modeling indicated that the rate of skin heating could be altered by assuming different levels of resting SkBF (Nelson et al. 2003). Taken together, these observations suggested that alterations in SkBF would alter the rate of skin heating in response to MMW exposure; this hypothesis was empirically tested in the current study.

Indeed, the rate of skin heating under LP-MMW exposure conditions was significantly affected by pre-exposure levels of SkBF (Fig. 2). Preheating the skin for 60 min raised SkBF approximately 1.5 to 2.0-fold; it should be noted that, in response to local heating, forearm SkBF may increase as much as 10 to 12-fold (Taylor et al. 1984). Despite the relatively mild increase observed in our study, SkBF had profound effects on the subsequent rate of skin heating. Inspection of Fig. 2 shows that (1) MMW heating substantially increased SkBF when the initial SkBF and  $T_{sk}$  were at normal levels; (2) the increase in SkBF correlated temporally

with the attainment of the plateau phase in the non-occluded forearm; and (3) in the absence of a rise in SkBF (i.e., in the occluded state),  $T_{sk}$  did not reach a plateau but continued to increase throughout exposure. Furthermore, when SkBF was occluded following attainment of this plateau phase,  $T_{sk}$  immediately increased rapidly; upon restoration of SkBF during LP exposure,  $T_{sk}$  rapidly returned to the plateau level (Fig. 3). These results indicate that attainment and maintenance of the steady-state plateau phase in  $T_{sk}$  during LP exposure is dependent on the presence of an increase in SkBF.

We have previously demonstrated that, under the conditions of brief (3–4 s) HP exposure, the rate of skin heating in animals (Nelson et al. 2003) and humans (Nelson et al. 2003; Walters et al. 2000) can be explained by a simple conduction model. In the current investigation, there were no differences in the rate of  $T_{sk}$  increase during HP exposure, despite a varied range of pre-exposure SkBF levels (Fig. 4). These observations further demonstrate that the rate of skin heating under these exposure conditions is adequately described by a one-dimensional thermal conduction model, and other avenues of heat transfer do not play a significant role.

The SkBF measurements in this study were limited to relative measurements, as it is not possible to directly measure blood flow (i.e., in  $\text{mL min}^{-1}$ ) using LDI. Thus, it is not possible to use these measurements to validate our previous simple model that included SkBF. Regardless, LDI did allow us to examine the interaction between SkBF and MMW-induced skin heating. From these data, it is evident that increased SkBF is the predominant mechanism in humans for heat transfer, even under the relatively high levels of MMW exposure that we defined as LP-MMW (for 94-GHz exposures, the IEEE C95.1 maximum permissible exposure for uncontrolled environments is  $10 \text{ mW cm}^{-2}$  over an averaging time of 39.3 s).

In the few instances in which humans would be exposed to relatively high levels of MMW (i.e.,  $175 \text{ mW cm}^{-2}$ ), it is clear that the SkBF response would provide adequate thermal protection, as it efficiently removed heat from the skin before thermal damage could occur. In this respect, it should be noted that extrapolations of data derived from animal studies for safety standard determination may be conservative in that, for longer duration exposures, the greater ability of humans to increase SkBF may act as a self-protective mechanism to reduce the possibility of thermal injury. In this regard, the relative magnitude of the increase in SkBF produced by preheating in this study was well below maximal levels, suggesting that there is a great deal of vasodilatory reserve available for additional heat transfer, if required. It should be noted that individuals differ in sex, fitness levels, and age, all of which might affect the SkBF

response to local heating (Havenith and van Middendorp 1990; Thomas et al. 1999; Havenith 2001). Our results are applicable to 94-GHz exposure; other frequencies with deeper or shallower penetration and with different absorption coefficients may show different responses.

## CONCLUSION

The magnitude of skin heating during MMW exposure of relatively long ( $>60$  s) duration is greatly influenced by both the initial level of SkBF and the ability of SkBF to increase during local heating in humans. Because of the increase in SkBF, increases in  $T_{sk}$  are limited during MMW exposures of this duration. In contrast, SkBF is not altered by relatively brief (4 s), high power MMW exposure, and surface temperature increases under these conditions can be described by a one-dimensional conduction model that neglects blood flow effects.

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